

# Respiratory diseases in wood workers

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**ABSTRACT** A survey was carried out on wood workers and on a group of unexposed, healthy controls. One group of wood workers (group A) were asymptomatic and another (group B) had symptoms either of chronic cough and dyspnoea on exertion (B1) or dyspnoea at work and bronchial hyperreactivity (B2). The control group (group C) was randomly selected from among a population of laboratory workers. No significant differences were found among the groups with respect to the frequency of atopy but the prevalence of a positive skin reaction to wood extracts was significantly higher in the asthmatic subjects. The adjusted FVC and FEV<sub>1</sub>/FVC% were significantly higher in B1 than in the other groups; the FEV<sub>1</sub> was lower in B1 than in B2 and in B2 than in the other groups; TL<sub>CO</sub> and K<sub>CO</sub> differed significantly in all four groups. A significant negative correlation was observed between FEV<sub>1</sub>, MEF<sub>50</sub>, TL<sub>CO</sub> and K<sub>CO</sub> and duration of exposure to wood dusts. The alveolar volumes were not significantly different between the groups and were not correlated with duration of exposure. These results confirm the observation that exposure to wood dust or to some bronchoreactive substances linked with wood working can induce chronic obstructive lung disease (COLD). Some cases of wood dust asthma seem to be related to an immediate allergic reaction, but precipitating antibodies appear to be an index of exposure rather than of disease. The decrease in diffusion capacity can be ascribed to a thickening of the alveolar capillary membranes secondary to an alveolitis like, non-symptomatic, allergic reaction.

Respiratory diseases due to the inhalation of wood dust have previously been described and have been attributed to mechanical irritation,<sup>1,2</sup> to pharmacological effects,<sup>3,4</sup> or to hypersensitivity mechanisms.<sup>5-9</sup> Clinical patterns of an immediate<sup>10-13</sup> or late<sup>7,13,14</sup> asthmatic reaction may be seen, or a combination of both.<sup>5-7,10,14,15</sup> Extrinsic allergic alveolitis has also been detected<sup>13,16,17</sup> among wood workers and reports have also been published of chronic obstructive respiratory effects.<sup>1,2</sup>

The aim of the present study was to assess the pathogenic mechanisms of non-malignant respiratory disease in wood workers.

## Materials and methods

A survey was carried out on wood workers employed in small furniture factories and on a group of healthy, unexposed subjects; the groups had the following characteristics (table 1):

*Group A*—Healthy, exposed.

*Group B*—Exposed, symptomatic.

*B1*—Chronic cough and dyspnoea (grade II) on exertion.

*B2*—Dyspnoea at work and bronchial hyper-reactivity.

*Group C*—Unexposed (randomly selected from laboratory population).

All subjects completed a standardised questionnaire<sup>18</sup> modified with questions concerning history of allergic disorders, family history of allergic disease, and relation of dyspnoea to job and work environment.

Skin prick tests were carried out with wood dust extract and with commercially available allergens (LOFARMA allergeni). All the skin tests were graded by following standardised criteria.<sup>19</sup>

Serological tests were performed to measure C3, C4, IgG, IgM, IgA, and IgE concentrations.<sup>19</sup> RAST<sup>20</sup> for extracts of woods from individual work environments were also performed and precipitating antibodies<sup>21</sup> against the same wood and against moulds were checked.

The following lung function tests were carried out:

Forced expiratory curves: (forced vital capacity (FVC), forced expiratory volume at one second (FEV<sub>1</sub>), maximal expiratory flow at 50% of expired, by means of a Collins Stead Wells computerised spirometer. The FEV<sub>1</sub>/FVC% ratio was computed from the raw data.

Table 1 Comparison of study groups means (SD in parentheses)

Groups	B1	B2	A	C	p
No	15	20	55	53	
Age (years)	47.9 (11.6)	47.0 (13.8)	46.2 (11.8)	47.8 (10.2)	NS
Height (m)	1.63 (0.04)	1.66 (0.07)	1.66 (0.08)	1.66 (0.07)	NS
Weight (kg)	67 (11.9)	67 (11.8)	68 (10.0)	71 (9.2)	NS
RG (c/ml)	5 (0.3)	4.9 (0.2)	5.1 (0.3)	4.9 (0.4)	NS
HB (g/dl)	13 (0.7)	14 (0.8)	13 (0.9)	14 (0.8)	NS
LE (years)	27.2 (13.2)	29.5 (15.7)	25.2 (13.2)	0.0 (0.0)	NC
Pack/year	12.2 (15.8)	8.3* (12.6)	13.4 (15.7)	12.4 (10.6)	< 0.05

NC = Not compared because one cell has zero value.

NS = F test in ANOVA  $p > 0.10$ .

\*Source of significant difference.

Single breath lung transfer factor for CO (TL<sub>CO</sub>), alveolar volume (AV), and diffusion coefficient (TL<sub>CO</sub>AV = K<sub>CO</sub>).

Non-specific provocation tests were also performed using carbacol cumulative dose or salbutamol challenge when the FEV<sub>1</sub> was less than 70% of the predicted value; responders were defined as having a 20% reduction/increase in FEV<sub>1</sub>.

All tests were performed following ATS standards.

Blood counts were also undertaken and the haemoglobin concentration measured.

#### STATISTICAL METHODS

The main characteristics of the four groups were compared by one way analysis of variance. Comparison of the qualitative variables among the groups was performed using chi-squared ( $\chi^2$ ) with no continuity correction.

To study the patterns of lung function variables in the groups, the effects of nuisance variables were removed by means of a multiple regression analysis, taking the lung function results separately as dependent variables, and height, weight, age, and smoking habits as predictive variables.

The adjusted values were computed, the individual residual values (observed—predicted) adding to the grand mean. The adjusted values were compared by

means of analysis of covariance (ANCOVA) in a general linear model, using type III SS for parameter estimation in an unbalanced design and with no empty cells. Comparisons between pairs of groups were performed using Sheffe's method for simultaneous multiple comparisons. All interaction effects were computed and their SS errors pooled if they had not reached the prefixed significance level.<sup>22</sup>

For all analysis, a critical nominal alpha = 0.05 was adopted.

#### Results

No significant differences were detected between the groups for the main characteristics (table 1) or for the C3, C4 concentrations. The concentrations for all classes of Ig were in the normal range. Ig precipitating antibodies against wood dust were detected in four of the exposed subjects (3 in group A, 1 in group B2) and in none of the controls. Only one subject in the B2 group showed an increase in specific IgE (for obeche dust) on RAST (class 2).

The data in table 2 show that no significant differences were found between the four groups with respect to the prevalence of reactivity to common allergens but there was in the prevalence of reactions to wood dusts. The prevalence of skin positivity to

Table 2 Prevalence of skin reactivity to (a) common allergens and (b) wood dust extracts

Groups	B1	B2	A	C
<i>Common allergens</i>				
Skin test	+	1	3	8
$\chi^2 = 6.52$ $p < 0.05$	—	14	17	47
<i>Wood dust extracts*</i>				
Skin test	+	0	9	3
$\chi^2 = 41.71$ $p < 0.0001$	—	15	11	52

\*Comparison between A and B2.

Table 3 Distribution of skin positivity to dust extracts of various species of woods (in exposed subjects)

Woods	Frequencies	
	Absolute	Relative %
Walnut	1	5.6
Obeche	7	7.8
Douglas	4	4.4
Mansonia	1	1.1
Chestnut	1	1.1
Framire	1	1.1
Abies	1	1.1
White poplar	2	2.2
Oak	1	1.1

Table 4 Comparisons of lung function variables with duration of exposure (LE) as covariate

Groups	FVC (l)	FEV <sub>1</sub> (l)	FEV <sub>1</sub> /VC	MEF <sub>50</sub> (l/s)	TL <sub>CO</sub> (mmol/m <sup>2</sup> KPa)	K <sub>CO</sub> (mmol <sup>2</sup> /m <sup>2</sup> KPa)	VA (l)
B1	3.71W	2.17W	0.574W	0.69W	6.80W	1.07W	6.23W
B2	3.99X	2.90X	0.719X	2.51X	7.07X	1.25X	5.68W
A	4.37X	3.25X	0.757X	3.11X	8.20Y	1.33Y	5.33Y
C	4.30X	3.35Y	0.771Y	4.47Y	9.63Z	1.74Z	5.74W
LE	0.0	-0.002	0.0	-0.021	0.0	0.0	0.0
LE <sup>2</sup>	0.0	-0.080	-0.001	-0.002	-0.003	-0.006	0.0
p <	0.05	0.001	0.001	0.001	0.01	0.001	NS

(1) Groups with the same letter are not significantly different ( $p > 0.05$ ).

(2) If B = 0.0, parameter not significantly different from 0 ( $p > 0.05$ ); otherwise  $p < 0.01$ .

(3) Dependent variables are adjusted for height, weight, age, and smoking habits.

wood extracts was significantly higher in asthmatic subjects than in the other groups. The frequency of reaction to various work is shown in table 3.

Among the adjusted lung function variables, FVC and FEV<sub>1</sub>/FVC% were significantly lower in B1 than in the other groups (B1 < B2 = A = C); the FEV<sub>1</sub> was lower in B1 than in B2 and in B2 than in the other group; for MEF<sub>50</sub> B1 < B2 < A = C (table 4). TL<sub>CO</sub> and K<sub>CO</sub> were significantly different between all groups (B1 < B2 < A < C). A significant negative correlation was detected between FEV<sub>1</sub>, MEF<sub>50</sub>, TL<sub>CO</sub>, and K<sub>CO</sub>, and duration of exposure to wood dusts (LE quadratic term). The AV was not significantly different between groups (B1 = B2 = A = C) and was not correlated with duration of exposure.

## Discussion

The results reported here confirm that exposure to wood dust or to bronchoactive substances related to wood working can cause chronic obstructive lung disease (COLD).<sup>1,2</sup> This is shown by the decrease in FEV<sub>1</sub>, FEV<sub>1</sub>/FVC%, and MEF<sub>50</sub> which is dependent on the square of the duration of exposure—that is, it is dose dependent.<sup>1,2</sup>

Some cases of wood dust asthma seem to be linked to an immediate allergic reaction, as shown by the higher prevalence of skin positivity to wood dust in the group of asthmatic subjects. This is in agreement with previous findings.<sup>5,6,8,11,23</sup>

The disagreement between serological and cutaneous tests may be explained by the pharmacological or irritant effects of the diagnostic extracts.<sup>19</sup> If this hypothesis were correct we would expect a random distribution of skin positivity among the four groups. On the contrary, however, skin reactivity was significantly linked with exposure and to the presence of bronchial asthma.

If the skin positivity is due to the presence of specific antibodies the disagreement between the prick

tests and the RAST could be explained by the presence of short term IgG,<sup>24</sup> not detectable with RAST, or by antigen degradation during the preparation of the paper disc.<sup>19</sup> It might also be because IgE is less permanent in the serum<sup>19</sup> than in the skin or because the antigenic determinant of wood dust is a non-protein fraction.<sup>12</sup>

Precipitating antibodies to wood dust seem to be linked to occupational exposure and not to respiratory disorders, as previously reported.<sup>15,19</sup>

One of the unexpected but important findings in this study was the occurrence of a significant reduction in diffusion capacity in the exposed as compared with the unexposed subjects; moreover in the exposed subjects the diffusion capacity was lowest in the asthmatics. The diffusion abnormalities indicate the presence of early parenchymal damage or impaired perfusion: anaemia or uneven distribution in pulmonary ventilation can be excluded by the lack of any difference in either haemoglobin or alveolar volume between the groups. Indeed, the diffusion capacity in the asthmatic subjects was measured during a symptom free period. The impairment in diffusion capacity may be related to a reduction in capillary blood volume due to the activity of vasoactive substances.<sup>13,25</sup> On the other hand, it may be ascribed to a thickening of the alveolar capillary membranes secondary to an alveolitis like,<sup>13,16,17</sup> non-symptomatic allergic reaction.

In conclusion, our findings show the importance of skin tests for detecting subjects allergic to wood dusts. The FEV<sub>1</sub> is confirmed as the test of choice for detecting effects on the lung because of its ability to distinguish between asthmatic and non-asthmatic subjects and its relation to the duration of exposure. Further studies are required to explain the pathogenesis of the reduction in TL<sub>CO</sub>.

## References

- Ashley MJ, Corey R, Chang Yeung M, McLean L, Maledy H, Grzybowski SO. A respiratory survey of cedar mill workers—II. *J Occup Med* 1978;20:328–32.

- 2 Chan-Yeung M, Lam S, Koener S. Clinical features and natural history of occupational asthma due to western red cedar. *Am J Respir Dis* 1982;3:411-15.
- 3 Chan-Yeung M, Ciglas PC, Henson PM. Activation of complement by plicatic acid, the chemical compound responsible for asthma due to western red cedar (*Thuja plicata*). *J Allergy Clin Immunol* 1980;65:333-57.
- 4 Evans E, Nicholls PJ. Hystamine release by western red cedar (*Thuja plicata*) from lung tissue in vitro. *Br J Ind Med* 1974;31:28-33.
- 5 Briatico-Vangosa G, Cardani A, Carosso A, et al. Asma professionale da polvere di triplochiton scleroxylon (obeche, samba). *Folia Allergologica Immunologica Clinica*. 1983;30:465-9.
- 6 Carosso A, Bugiani M, Romano C. Asma professionale da polvere di obece (triplochiton scleroxylon). *Atti Del Convegno Nazionale—Infortuni e malattie professionali nel settore del legno e del mobile—SIENA 28 May 1982*. Vol 2. Siena: Amministrazione Provinciale Di Siena, 1983:464-9.
- 7 Chan-Yeung M. Maximal expiratory flow and airway resistance induced bronchoconstriction in patients with asthma due to western red cedar (*Thuja plicata*). *Am Rev Respir Dis* 1973;108:1103-10.
- 8 Nava C, Brambilla G, Briatico Vangosa G, Marchisio M. La immunoterapia specifica contro essenza di legno. *Med Lav* 1978;3(suppl):463-70.
- 9 Tze KS, Chan H, Chan-Yeung M. Specific IgE antibodies in workers with occupational exposure to western red cedar. *Clin Allergy* 1982;12:249-58.
- 10 Both BH, Le Foldt RH, Moffit EM. Wood dust hypersensitivity. *J Allergy Clin Immunol* 1976;57:352-7.
- 11 Innocenti A, Angotzi G. Asma professionale da sensibilizzazione a Tryplochiton scleroxylon (samba, obece). *Med Lav* 1980;3:251-4.
- 12 Paggiaro PL, Cantalupi R, Filieri M, et al. Bronchial asthma due to inhaled wood dust: Tanganika Anigree. *Clin Allergy* 1981;111:605-10.
- 13 Sosman AJ, Shlueter DP, Fink JM, Barboriak JJ. Hypersensitivity to wood dust. *N Engl J Med* 1969;281:977-80.
- 14 Chan-Yeung M, Abboud R. Occupational asthma due to California red wood (*sequoia sempervirens*) dusts. *Am Rev Respir Dis* 1976;114:1027-31.
- 15 Bush RK, Yunginger JW, Reed CE. Asthma due to African Zebrawood (*Microberlinia*) dust. *Am Rev Respir Dis* 1978;117:601-3.
- 16 Belin L. Clinical and immunological data on "wood trimmer disease" in Sweden—with discussion. *Eur J Respir Dis* 1980;61(suppl 107):169-76.
- 17 Howie AD, Boyd G, Moran F. Pulmonary hypersensitivity to Ramin (*Gonistylus Balcanus*). *Thorax* 1976;31:585-7.
- 18 BMRC Committee on the Aetiology of Chronic Bronchitis. Standardized questionnaires on respiratory symptoms. *Br Med J* 1960;ii:1665-80.
- 19 Both BH. Diagnosis of immediate hypersensitivity. In: Patterson R, (ed). *Allergic diseases*. Philadelphia: JB Lippicott, 1980:63-76.
- 20 Berg TLO, Johansson SGO. Allergy diagnosis with the radio-allergosorbent test (RAST). *J Allergy Clin Immunol* 1974;54:209-15.
- 21 Outcherlony D. Antigen-antibody reaction in gels: types of reactions in coordinated systems of diffusion. *Acta Pathol Microbiol Scand* 1953;32:231-70.
- 22 Weinsberg S. *Applied linear regression*. New York: John Wiley & Sons, 1980.
- 23 Greemberg M. Respiratory symptoms following brief exposure to cedar of Lebanon (*Cedrea Libani*) dust. *Clin Allergy* 1971;2:219-21.
- 24 Parish WE. Some biological activities of IgG subclass antibodies after inoculation and in disease. In: Ganderton MA, Frankland AW, eds. *Allergy*. Tunbridge Wells: Pitman Medical, 1975.
- 25 Zafriropulo A, Audibert A, Charpin J. A propos des accidents dus à la manipulation des bois exotiques. *Revue Française d'Allergologie* 1958;8:155-71.